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Termination of Ventricular Tachycardia With Epicardial Laser Photocoagulation: A Clinical Comparison With Patients Undergoing Successful Endocardial Photocoagulation Alone

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Electrical activation-guided laser photocoagulation was used intraoperatively to terminate ventricular tachycardia in patients with ischemic heart disease. During ventricular tachycardia, laser irradiation was delivered to mapped sites with local diastolic activation. In 30 long-term survivors, 85 ventricular tachycardia configurations were terminated by ablation; 72 (84.7%) were terminated by endocardial photocoagulation. Thirteen (15.3%) required epicardial photocoagulation; however, these 13 ventricular tachycardias occurred in 10 (33%) of the 30 patients. An aneurysm was present in 70% of patients with successful endocardial photocoagulation, but in only 10% of patients requiring epicardial photocoagulation for at least one ventricular tachycardia configuration; 90% of all patients requiring epicardial laser photocoagulation had no aneurysm and had either a right or a left circumflex coronary artery-related infarction.

In this group, epicardial activation data were similar to those described for ventricular tachycardia with an "endocardial" origin and included 1) delayed potentials during sinus rhythm, 2) presystolic or pandiastolic activation sequences during ventricular tachycardia, and 3) regions of block near the presumed region of reentry during ventricular tachycardia. This study suggests that the critical anatomic substrates supporting reentry in postinfarction ventricular tachycardia may occur at intramural or epicardial sites, particularly in patients with right or circumflex coronary artery-related infarction and no aneurysm.

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Myocardial laser photocoagulation using the continuous wave 1.06 μm neodymium:yttrium-aluminum-garnet (Nd:YAG) laser has been a successful method for the intraoperative ablation of ventricular tachycardia (1–3). Laser photocoagulation of the myocardium is performed during ventricular tachycardia. This provides a unique opportunity to take a strictly empiric approach in correlating intraoperative mapping data with the site of laser photocoagulation terminating ventricular tachycardia. Observations derived from the ablation of 85 tachycardia configurations in 35 patients have forced us to suspend certain judgments regarding the critical role of the endocardium or subendocardium in all ventricular tachycardias complicating previous myocardial infarction.

The purpose of this study was to analyze the intraoperative mapping data from those patients in whom epicardial laser photocoagulation was necessary to terminate ventricular tachycardia and compare their clinical features with those patients in whom endocardial irradiation alone was successful.

Methods

Study patients. Between January 1, 1985 and January 1, 1988, 35 patients with previous myocardial infarction underwent intraoperative laser photocoagulation of ventricular tachycardia. This study analyzed the results in 30 of the 35 patients surviving the procedure for >3 months. All patients had ventricular tachycardia refractory to pharmacologic agents as judged by spontaneous recurrences or electrophysiologic studies, or both. In addition to preoperative electrophysiologic study, all patients underwent electrophysiologic

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study before hospital discharge and at 3 months and 1 year postoperatively. Exceptions were three patients who refused electrophysiologic study at 3 months and four patients who refused restudy at 1 year. Telephone and written contact has been maintained on a routine basis since the time of discharge. All patients signed informed consent to participate in the research study approved by the U.S. Food and Drug Administration and the Research and Human Subjects Protection Committee of Charlotte Memorial Hospital and Medical Center.

Operative selection. No patient was excluded from surgery on the basis of 1) infarct location, 2) presence or absence of an aneurysm, 3) number of ventricular tachycardia configurations, or 4) characteristics of ventricular tachycardia. Refractory ventricular tachycardia was a sole indication for surgery except in two patients in whom coronary bypass surgery was also independently indicated. A wall segment was considered aneurysmal if it was dyskinetic, collapsed when the left ventricle was vented and was ≤5 mm in thickness. A ventriculotomy was performed in all patients whether or not an aneurysm was present. In patients with right coronary artery-related infarction, the ventriculotomy was performed between the septum and the posterior papillary muscle. In patients with circumflex coronary artery-related infarction, the ventriculotomy was made between the papillary muscles.

Intraoperative electrophysiologic studies. Surface electrocardiographic leads I, II and III, with or without V_6, were recorded with an epicardial reference electrode. Mapping data were obtained using a hand-held probe recording both unipolar (filtered 0.5 Hz to 1 kHz) and bipolar (filtered 50 Hz to 1 kHz) data. These date were recorded on a Siemens Mingograf recorder at a paper speed of 250 mm/s. Immediate timing of the local activation sequence was facilitated by displaying the limb leads, epicardial reference and unipolar and bipolar mapping data on an eight channel Tektronix oscilloscope triggered from the QRS complex or reference electrode during ventricular tachycardia. This permitted easily reproducible, immediately accessible discrimination of activation timing. During this study, attempts were not made to acquire global endocardial or epicardial activation sequences. Instead, attention was focused on endocardial or epicardial regions, or both, with local activation beginning at the end of one QRS complex to the onset of the next QRS complex during ventricular tachycardia. This system of mapping allowed a spatial resolution of 5 mm. It was hypothesized that laser photocoagulation of part or all of this region would be required to terminate ventricular tachycardia. Ventricular tachycardia was initiated with trains of rapid stimuli or premature stimulation before or after ventriculotomy, or both. Particular attention was directed to maintaining a normothermic myocardium.

Intraoperative laser photocoagulation. The laser source was a continuous wave Nd:YAG laser (λ = 1.06 μm) coupled to a 600 μm gas-cooled silica quartz fiber. In our initial experience, a standard fiber was used with a 10° beam divergence angle, using a radiating spot size of approximately 0.5 cm. This required a fiber-tissue working distance of approximately 2 cm. Certain practical limitations of this delivery system imposed by anatomic constraints and geometric inhomogeneities of the surface led us to develop a 20° beam divergence fiber (MBB-Medizintechnik) used in the last 11 cases (4). Irradiating powers of 30 to 50 W were used with the 10° divergence fiber and 60 to 80 W with the 20° divergence fiber. The irradiated surface was kept free of blood and, at times, the surface was cooled to enhance depth of penetration. With use of this method of laser irradiation, the maximal depth of myocardial photocoagulation is usually 5 to 6 mm (1,2).

The initial site chosen for laser photocoagulation displayed local activation in mid-diastole during ventricular tachycardia. If such a site could not be identified, the site showing the earliest presystolic activity was chosen. If laser irradiation at the initial site failed to terminate ventricular tachycardia, the irradiated area was enlarged to include other contiguous areas with diastolic electrical activity. Laser irradiation was delivered in this manner to the endocardium or epicardium until ventricular tachycardia terminated and could no longer be reinduced. If laser irradiation was not successful in terminating ventricular tachycardia, appropriate epicardial or endocardial sites were remapped and laser irradiation was delivered to other sites as indicated by mapping data.

All electrical activation mapping data and laser irradiation data were referenced to a grid drawing of the endocardium and epicardium of the right and left ventricular myocardium (Fig. 1). The irradiated surface area was measured for calculation of energy density (joules/cm^2) of delivered laser irradiation. The duration of irradiation at each site was available by an automatic printout from the MediLas 2 Nd:YAG laser instrument.

Postoperative electrophysiologic studies and follow-up. All 30 patients underwent predischarge electrophysiologic study using epicardial wires placed at surgery (18 patients), catheter stimulation from two right ventricular sites (8 patients) or catheter stimulation from one right ventricular endocardial site by a permanent pacemaker that could be externally coupled to a programmed stimulator (4 patients). In each case, programmed stimulation up to S_4 at two cycle lengths and pacing trains of 8 to 12 stimuli to a cycle length resulting in loss of 1:1 capture were employed. Postoperative studies were also performed at 2 to 3 months and at 1 year. Endocardial stimulation as just described was employed at two right ventricular sites, except in four patients in whom an externally coupled permanent pacemaker was used.

A detailed analysis of the acquired intraoperative mapping and lasing data was performed in 10 of the 30 surviving patients in whom the following criteria were met: 1) mapping

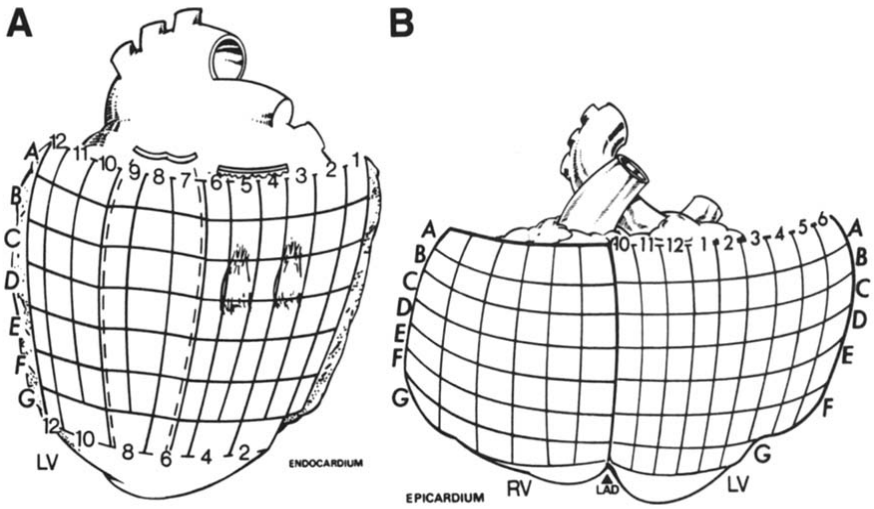

Figure 1. The reference grid for endocardial (A) and epicardial (B) mapping sites. The endocardial surface (A) is divided into concentric rings A through G from the base to apex. Each ring has 12 points (except G, which has 6). Left ventricular epicardial sites (B) correspond to the underlying endocardial sites. Laser irradiation was delivered and referenced to the same grid. LAD = left anterior descending coronary artery; LV = left ventricle; RV = right ventricle.

data during ventricular tachycardia demonstrated either presystolic or pandiastolic activation sequences on the epicardium, and 2) laser photocoagulation of the epicardium terminated ventricular tachycardia either before or after unsuccessful endocardial irradiation. In these patients, all epicardial local activation data were analyzed in terms of timing during diastole and unipolar and bipolar electrogram characteristics. Careful observations were made on the size of the area with diastolic potentials during ventricular tachycardia and the measured irradiated area. This group was compared with the 20 patients with successful endocardial laser photocoagulation alone with respect to clinical variables. The variables included infarct-related coronary vessels and presence or absence of an aneurysm. All data are expressed as mean values ± SD.

Results

Surgical results. The overall hospital mortality rate was 14%. Table 1 presents the incidence of death as a function of two clinical variables, namely, ejection fraction and presence of an aneurysm. In patients with an ejection fraction <20% and no aneurysm, the mortality rate was 66.7% (two of three patients). For patients with an ejection fraction <20% but with a discrete left ventricular aneurysm, the mortality rate was 28.5% (two of seven patients). However, for patients with an ejection fraction ≥20% with or without an aneurysm, the mortality rate was only 4% (1 of 25 patients). Preoperative left ventricular function seemed to be an important determinant of survival.

Clinical results. Electrophysiologic studies were performed before discharge, at 2 to 3 months after discharge and at 1 year. Table 2 lists the clinical results in the 30 patients discharged from the hospital. The mean follow-up period is 20.1 ± 9.3 months. There has been no spontaneous recurrence of ventricular tachycardia up to 2 years postoperatively. One of the 14 patients surviving for >2 years had recurrence of ventricular tachycardia at 26 months induced by rapid atrial fibrillation during acute congestive heart failure. One patient had inducible sustained ventricular tachycardia before discharge. An automatic implantable cardioverter/defibrillator was implanted, with no spontaneous recurrence up to 18 months. Three patients had inducible nonsustained ventricular tachycardia of 5 to 21 beats. At 3 months, tachycardia was no longer inducible in one of these three patients, one patient continued to have nonsustained ventricular tachycardia and one developed inducible

Table 1. Hospital Deaths in 35 Patients

EF %	Aneurysm	No. of Patients
<20	0	2 of 3 (66.7%)
<20	+	2 of 7 (28.5%)
≥20	0/+	1 of 25 (4%)

EF = ejection fraction; 0 = no aneurysm; + = aneurysm present; 0/+ = without or with aneurysm.

Table 2. Functional Anatomy and Lased Surface in Patients Undergoing Photocoagulation

Infarct Vessel		Total	Lased Surface: Endo	Lased Surface: Endo (no VT)	Lased Surface: Epi
LAD	16				
Aneurysm		12 (75%)	7	4	1
No aneurysm		4	3	1	0
LCx/RCA	14				
Aneurysm		3 (21%)	3	0	0
No aneurysm		11	2	0	9
Total	30		15	5	10

Endo = endocardium; Epi = epicardium; LAD = left anterior descending artery; LCx = left circumflex artery; no VT = no inducible ventricular tachycardia at surgery; RCA = right coronary artery.

sustained ventricular tachycardia. The latter patient refused an automatic implantable cardioverter/defibrillator and has remained free of tachycardia recurrence for 12 months. Twenty-three patients have been followed up for at least 1 year. Of these, 19 agreed to repeat electrophysiologic study. No patient had inducible sustained ventricular tachycardia. Only one patient had inducible nonsustained ventricular tachycardia (nine beats); this patient had spontaneous recurrence of tachycardia at 26 months.

Clinical correlates (Table 2). The 30 surviving patients were analyzed in relation to the infarct-related vessel, the presence or absence of a discrete left ventricular aneurysm and the need for endocardial irradiation or epicardial laser irradiation to terminate all configurations of ventricular tachycardia at surgery. Sixteen patients had left anterior descending and 14 patients had right or circumflex coronary artery-related infarction. An aneurysm was present in 12 (75%) of the 16 patients with left anterior descending artery-related infarction, but in only 3 (21%) of the 14 patients with right or circumflex coronary artery-related infarction. In five patients, ventricular tachycardia could not be initiated at surgery; all five had a left anterior descending artery-related infarction and four (80%) of the five had a discrete left ventricular aneurysm. Ventricular tachycardia could not be initiated in 4 (33%) of 12 patients with an aneurysm due to left anterior descending artery-related infarction, but in only 1 (5.6%) of the 18 remaining patients. In the five patients with no inducible ventricular tachycardia at surgery, laser photocoagulation was confined to the endocardium and was both visually directed and guided by preoperative catheter mapping studies. The 25 patients with inducible ventricular tachycardia at surgery had a total of 78 ventricular tachycardia configurations (mean 3.12 ± 1.42 per patient, range 1 to 6 per patient).

Epicardial versus endocardial laser photocoagulation. To be included in the group having epicardial laser photocoagulation, patients had to have a stable, reproducibly initiated ventricular tachycardia that was convincingly terminated during epicardial laser photocoagulation and could not be reinduced subsequently. Thirteen (15.3%) of the 85 configurations at surgery fulfilled these criteria. The 13 ventricular tachycardias terminated by epicardial photocoagulation occurred in 10 (33%) of the 30 patients. There were 11 patients with right or circumflex coronary artery-related infarction and no aneurysm. Nine (82%) of these 11 patients required epicardial laser photocoagulation to terminate at least one of the ventricular tachycardias present.

A total of 10 patients required epicardial laser photocoagulation. In this group, there were 36 configurations of ventricular tachycardia; however, only 13 (36%) of the 36 required epicardial ablation. The remainder were successfully terminated by endocardial photocoagulation. All patients in this group had at least one ventricular tachycardia configuration requiring endocardial photocoagulation. In 6 of the 10 patients, epicardial mapping and epicardial laser photocoagulation were performed only after endocardial laser photocoagulation proved ineffective. However, in four patients, epicardial laser photocoagulation alone was performed for that particular ventricular tachycardia. All three patients with an aneurysm associated with right or circumflex coronary artery-related infarction underwent successful ablation with endocardial laser photocoagulation alone, as did 11 (92%) of 12 patients with an aneurysm associated with left anterior descending artery-related infarction.

Electrical activation phenomena observed on the epicardium. For patients in whom all ventricular tachycardia configurations were terminated by endocardial laser photocoagulation, epicardial mapping data were not consistently obtained. Epicardial electrograms were uniformly obtained in the 10 patients requiring epicardial laser photocoagulation. Electrical activation phenomena previously described on the endocardial surface were found on the epicardial surface in this patient subgroup. These included delayed potentials during sinus rhythm, diastolic potentials during ventricular tachycardia that, at times, could be recorded from the end of one QRS complex to the onset of the next QRS complex and low amplitude diastolic potentials during ventricular tachycardia with various degrees of block.

Figure 2A illustrates endocardial potentials during sinus rhythm recorded between the posterior papillary muscle and the mitral anulus and local activation on the directly opposite epicardial surface. A delayed potential 4 ms after the end of the QRS complex is recorded from the endocardium, but 68 ms after the end of the QRS complex from the epicardial surface. In Figure 2B, during ventricular tachycardia unipolar recordings from the same epicardial location registered a negative QS pattern 8 ms before the onset of the QRS complex. Laser photocoagulation directed at this epicardial site and toward the septum terminated ventricular tachycardia.

Figure 3 illustrates pandiastolic activity during ventricular tachycardia recorded on the epicardial surface from the end of one QRS complex to the onset of the next QRS complex. Diastolic activation proceeded from the left side of the posterior interventricular groove near the interventricular septum to the right side of the interventricular groove near the apex. The total length of this segment was 8 to 10 cm. Laser irradiation of this epicardial surface (shaded area) permanently abolished this ventricular tachycardia.

Figure 4 illustrates a configuration of ventricular tachycardia in another patient in whom potentials could be recorded from mid-diastole to the onset of the next QRS complex on the inferior right ventricular free wall. This patient had a right coronary artery-related infarction, with a patch of fibrosis visible on the epicardial surface of the inferior right ventricle. Three other ventricular tachycardia configurations had been terminated by endocardial laser photocoagulation of the left ventricle. The diastolic poten-

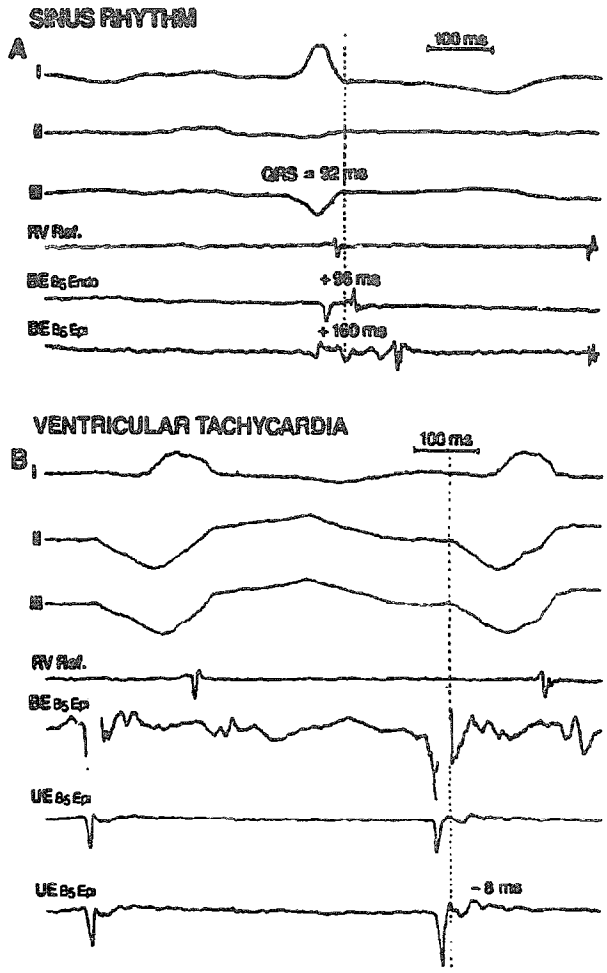

Figure 2. A, Bipolar electrograms (BE) recorded from the endocardial (endo) and opposite epicardial (epi) surface during sinus rhythm. B, During ventricular tachycardia, the epicardial region of marked delay during sinus rhythm activated 8 ms before the onset of the QRS complex, with the unipolar electrograms (UE) displaying a QS pattern. Dotted line denotes onset of the QRS complex. Laser irradiation of this epicardial region terminated ventricular tachycardia. RV Ref = right ventricular reference tracing.

tials illustrated were recorded 1 to 2 cm from the posterior interventricular groove. Ventricular tachycardia was terminated by laser photocoagulation of the shaded area (Fig. 4B).

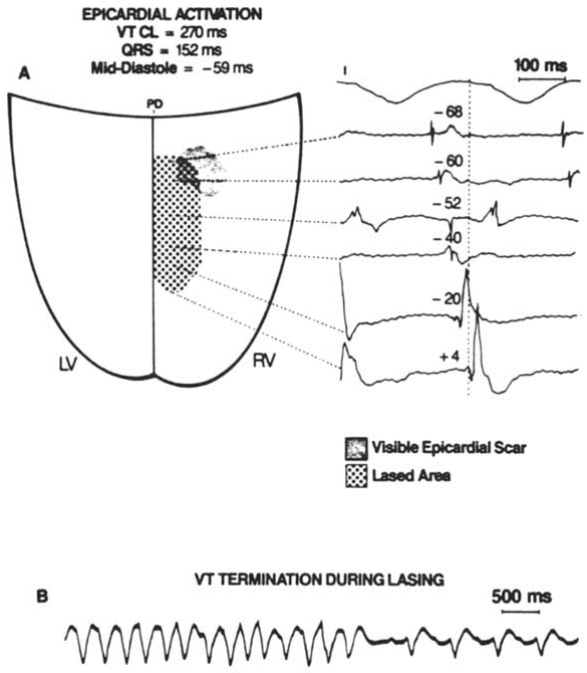

Figure 4. A, The location of bipolar electrograms recorded during ventricular tachycardia, with local activation from before mid-diastole to the onset of the QRS complex. The shaded area denotes visible scar. The stippled area shows the laser-irradiated area. B, Termination of ventricular tachycardia (VT) during laser photocoagulation of this region. Abbreviations as in Figures 2 and 3.

Figure 5 illustrates endocardial and epicardial potentials recorded in a patient with successful epicardial laser ablation of ventricular tachycardia. Epicardial diastolic activation begins 30 ms after the end of the QRS complex on the inferior wall between the anterior and posterior papillary muscles. Successively later potentials are recorded toward the posterior interventricular groove until potentials 88 ms before the QRS complex are recorded over the septum in a plane between the papillary muscles and mitral anulus (mid-diastole at −102 ms). On the endocardial surface, lower amplitude and more slurred potentials are recorded at 80 ms before the onset of the QRS complex at the junction between the septum and the inferior wall in a plane between the mitral anulus and the papillary muscles. From that point, activation

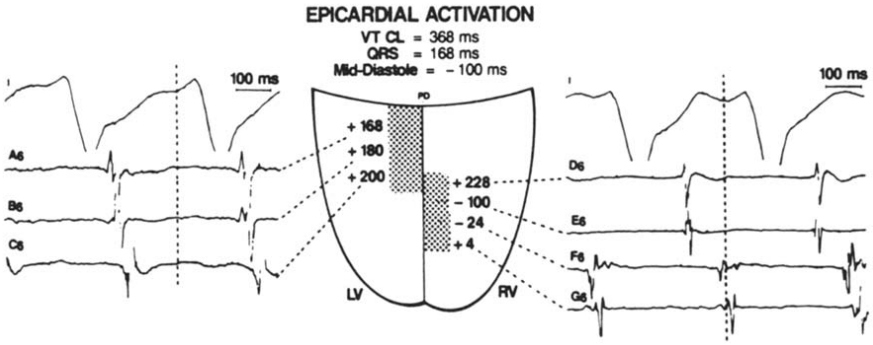

Figure 3. Epicardial pandiastolic activation recorded from the end of one QRS complex to the onset of the next QRS complex. Local activation times progressed along the inferior surface of the left ventricle (LV) adjacent to the posterior descending coronary artery (PD) and then to the inferior surface of the right ventricle (RV) toward the apex. Laser photocoagulation of the shaded area terminated the arrhythmia. VT CL = ventricular tachycardia cycle length.

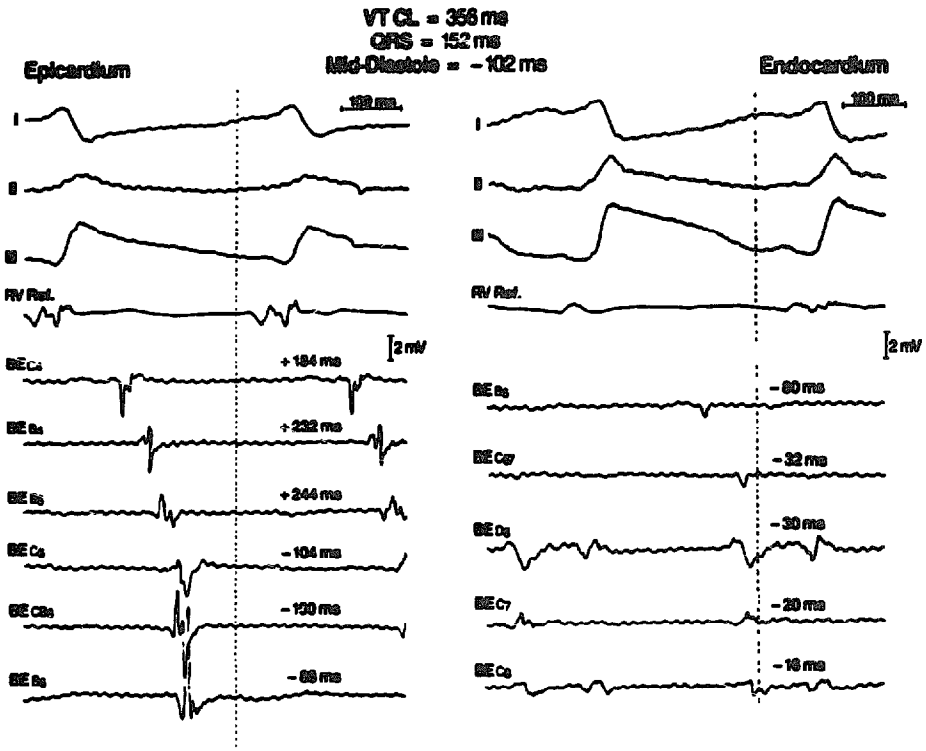

Figure 5. During ventricular tachycardia, bipolar electrograms with local activation in diastole were recorded from both the epicardium (**left panel**) and endocardium (**right panel**). **Dotted line** denotes onset of the QRS complex. On the epicardial surface, local activation is recorded from 37 ms after the end of the QRS complex to 88 ms before the onset of the QRS complex. On the endocardial surface, potentials are recorded from 80 ms after the end of the QRS complex to 16 ms before the onset of the QRS complex. Laser photocoagulation of these endocardial sites failed to terminate the arrhythmia. However, laser photocoagulation of the epicardial sites did terminate the ventricular tachycardia. Abbreviations as in Figures 2 and 3.

times progress closer to the onset of the QRS complex at the mid-portion of the interventricular septum. Laser photocoagulation of these endocardial sites failed to terminate the ventricular tachycardia. However, laser photocoagulation of the epicardial sites permanently eliminated the arrhythmia.

Figure 6 illustrates a region of block closely adjacent to a region of presumed reentry. These recordings are from the only patient with an anterior infarction in whom epicardial laser photocoagulation was necessary to terminate one of the ventricular tachycardias. In Figure 6A, the stippled area indicates the epicardial region of recorded potentials and the area of laser photocoagulation. Figure 6B illustrates markedly delayed epicardial potentials during sinus rhythm recorded from this region. In Figure 6C, a unipolar electrogram over a region of scar was used to help identify the onset of the cavitylike potential for activation time measurements (5). Epicardial potentials were recorded from 132 ms after the onset of the QRS complex up to 40 ms before the onset of the next QRS complex. In a closely adjacent region, 2:1 block of the electrograms is recorded during the diastolic phase of ventricular tachycardia (Fig. 6D). Epicardial laser photocoagulation successfully terminated this ventricular tachycardia.

Discussion

Background: epicardial versus endocardial surgical approaches. Surgical ablation strategies for ventricular tachycardia were initially directed toward the epicardium. This approach was based in part on the success of epicardial map-guided surgical ablation of aberrant pathways in the Wolff-Parkinson-White syndrome. Early epicardial map-guided approaches to ventricular tachycardia ablation included cryothermia (6,7) and surgical incision or resection through areas of delayed activation during sinus rhythm or areas with diastolic activation during ventricular tachycardia (8–10). Although some success was achieved with these early epicardially directed approaches, the results were not encouraging when applied to the broad spectrum of patients with postinfarction ventricular tachycardia.

In this setting, the surgical cure rates achieved with map-guided endocardial resection for postinfarction ventricular tachycardia defocused attention from the potential role of intramural or epicardial sites of origin (11). The importance of the epicardial region was further deemphasized by reported discrepancies (12) between endocardial and epicardial activation sequences during ventricular tachycardia. These studies (11,12) suggested that epicardial mapping may fail to disclose the true site of origin of ventricular tachycardia.

Other endocardially directed approaches were developed. These included visually directed resection of all scarred endocardial surface (13), visually directed complete encircling endocardial ventriculotomy (14), map-guided and visually guided partial encircling endocardial ventriculotomy (15), cryothermal and Nd:YAG thermal exclusion of the site of origin of ventricular tachycardia (16), argon laser volatil-

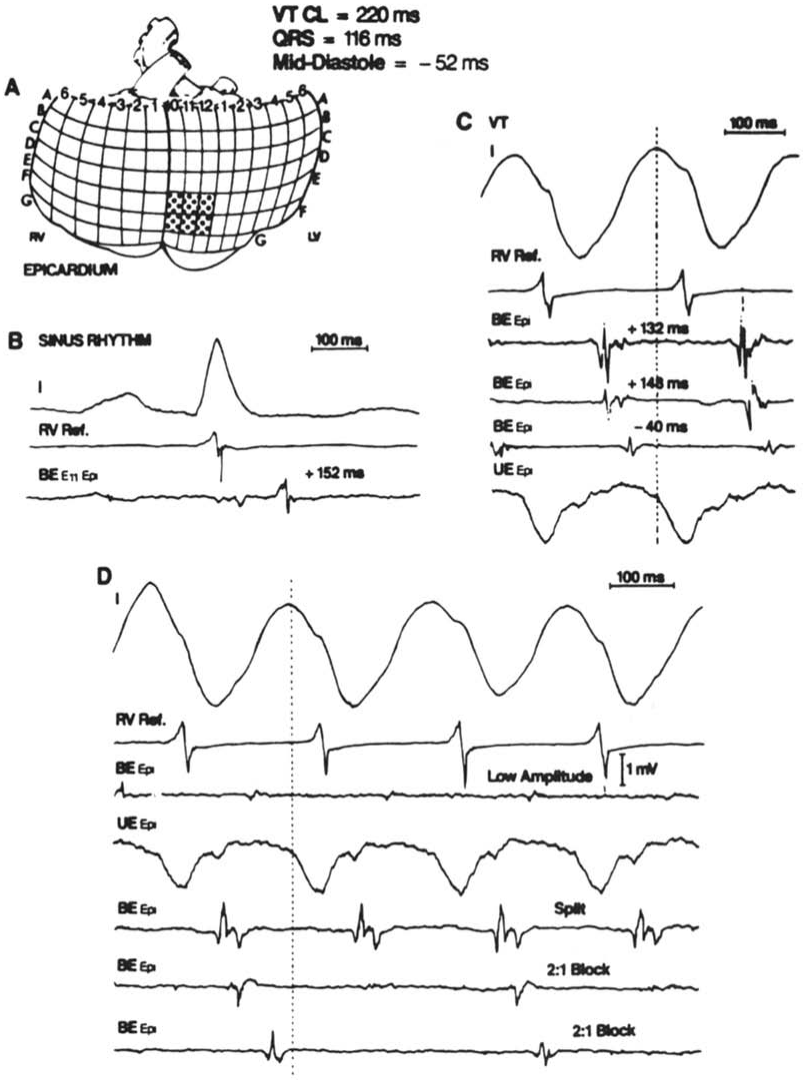

Figure 6. A region of block closely adjacent to a region of presumed reentry. A, The stippled epicardial (epi) area shows the region of recorded potentials and the laser-treated area. Delayed potentials during sinus rhythm could be recorded from this area (B). During ventricular tachycardia (VT) (C), diastolic potentials could be recorded up to 40 ms before the onset of the QRS complex. In a closely adjacent area, 2:1 block was recorded at several sites (D). Abbreviations as in Figures 2 and 3.

ization of the endocardial surface (17), and transmural septal (18) and extensive endocardial (19) cryoablation.

From these endocardially directed approaches, two empiric observations suggested that the critical anatomic substrates of postinfarction ventricular tachycardia were not always in a subendocardial location. Endocardially directed approaches have a significantly higher failure rate in patients without a discrete left ventricular aneurysm and in patients with an inferior or a posterior infarction (20). Also, deep cryoablation, even transmural cryoablation, has been required to ablate some ventricular tachycardias (18,19). Furthermore, recent mapping studies (21) in postinfarction ventricular tachycardias using plunge needle electrodes have suggested intramural reentrant circuits.

Current observations. In this context, our observations derived from ventricular tachycardia termination by photocoagulation during ventricular tachycardia are of interest. Mapping data and sites of successful laser ablation of ventricular tachycardia were analyzed with respect to the clinical variables known to be risk factors for surgical failure using endocardially directed approaches. The two most widely recognized risk factors for surgical failure are the absence of a discrete left ventricular aneurysm and the presence of an inferoposterior infarction (20). Of the 10 patients requiring epicardial laser photocoagulation, 9 had infarction in the distribution of the right or circumflex coronary artery. Of the 11 patients with a right or circumflex coronary artery-related infarction without an aneurysm, 9 (88%) required epicardial laser photocoagulation to terminate at least one ventricular tachycardia. In contrast, of the 20 patients in whom all ventricular tachycardias were terminated by endocardial photocoagulation, a left anterior descending artery-related infarction was present in 15 (75%) and a discrete aneurysm was present in 14 (72%). Endocardial photocoagulation alone was successful in 92% of the 14 patients with an aneurysm, but would have been unsuccessful in 60% of the 15 patients without an aneurysm. Epicardial electrical activation phenomena in patients with epicardial

laser ablation were similar to those described on the endocardial surface, including 1) delayed potentials during sinus rhythm, 2) pandiastolic or presystolic activity during ventricular tachycardia, and 3) regions of block near the reentrant circuit.

Anatomic correlations. Anatomic observations during surgery were of interest. In patients without an aneurysm, particularly those with a right or a circumflex coronary artery-related infarction, endocardial fibrosis was patchy. In fact, there was no discrete plane of fibrosis that could have been resected. Patchy fibrosis was frequently present on the epicardial surface as well. These findings suggest that the critical anatomic substrates of ventricular tachycardia are not in a uniform region of the three-dimensional structure of the myocardium and may be influenced by the absence of an aneurysm and the infarct location. We do not suggest that all ventricular tachycardias arise from intramural or epicardial sites. Only 13 (15.3%) of the 85 ventricular tachycardia configurations required epicardial laser ablation. However, an epicardial approach was required in 10 (33%) of 30 patients when operative selection did not exclude patients with inferior infarction or patients without an aneurysm. An effective ablation technique will need to be adaptable to a multiplicity of locations within the three-dimensional cardiac structure.

Conclusions. The results of map-guided Nd:YAG laser photocoagulation suggest that, in certain cases, the critical anatomic substrates of reentry in postinfarction ventricular tachycardia may be intramural or epicardial. This condition is more likely to occur in patients without an aneurysm and with right or circumflex coronary artery-related infarction. Application of Nd:YAG laser photocoagulation can be effective independent of the anatomic site of reentry and may be useful in extending our understanding of this complex electrophysiologic-anatomic problem.

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